## LETTER TO THE EDITOR

# Treatment of Atypical Postparturient Udder Edema in Goats

#### DEAR SIR:

Further to the previous discussion (Can Vet J 1983; 24: 62) of the vitamin-mineral-hormone interrelationship operating prior to development and during the clinical syndrome in goats of atypical postparturient udder edema (APPUE). Observations from several additional references may be of value when critically appraising the effectiveness of the components presented in a commercial product presently administered for the treatment of APPUE in goats.

Many biochemical reactions occurring within metabolic pathways are controlled by the magnesium concentration and the rate of initiation and maintenance of calcium mobility may be slowed when magnesium concentrations are low (9). The nucleotide, cyclic adenosine monophosphate (cAMP), is produced from ATP through the action of the enzyme, adenylate cyclase, and this enzyme requires magnesium for activation (4). Magnesium has been shown to be necessary for the release of parathyroid hormone from the parathyroid. gland (1,8). Magnesium deficiency inhibits the action of parathyroid hormone on calcium mobility from bone and phosphorus excretion from kidneys (5). The first hydroxylation of vitamin D to 25-hydroxycholecalciferol occurs in the liver and requires magnesium. The second hydroxylation to 1,25 dihydroxycholecalciferol occurs in the kidney and requires parathyroid hormone as a trophic hormone (3). Subclinical hypomagnesemia reduced the ability of dairy cows to mobilize calcium in response to hypocalcemia (9). The availability of magnesium may be reduced by the excessive dietary intake of calcium and phosphorus (9). When dietary calcium is increased the dietary magnesium requirement is also increased (6). Both calcium and phosphorus are absorbed by vitamin D-dependent pathways (11). A high serum phosphate level, associated with a magnesium deficiency, selectively precipitates out the

calcium with deposition into soft tissues (7). Dietary phosphate appears to be more important than calcium in aggravating magnesium deficiency (7). The effect of hypomagnesemia may be produced by the interference with the secretion of parathyroid hormones, by interference with this hormone on its target organs or by interfering with the metabolism of vitamin  $D_3$  (2,10).

Assessment of the above observations, as well as those reported in Can Vet J 1983; 24: 62, with regard to the clinical syndrome-APPUE in goats, which occurs under management conditions previously described in Can Vet J 1982; 23: 275, would appear to indicate that the therapeutic response to the intramuscular injection of the commercial product is due solely to vitamin D<sub>3</sub>.

Therefore, for prevention of APPUE in goats, reiteration of the previously advocated preventative-animal-health recommendations appear to be most apropos.

Sincerely yours, J.A. MILLS, D.V.M. 2211 West 37th Avenue Vancouver, B.C. V6M 1P2

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### **ABSTRACT**

CHAY S, WOODS WE, NUGENT T, BLAKE JW, TOBIN T. The pharmacology of nonsteroidal antiin-flammatory drugs in the horse: flunixin meglumine (Banamine). Equine Practice 1982; 4: 16-23. (Dept. Vet. Sci., Coll. Agric., Univ. Lexington, Kentucky 40546).

After rapid i/v injection of 1 mg/kg of flunixin, plasma levels of the drug peaked at  $10 \mu g/ml$  three minutes after injection of the drug. Thereafter, plasma levels of the drug fell rapidly, with an apparent alpha phase half-life of about 11.8 minutes. This was followed by a slower beta phase halflife of about 1.6 hours. Evidence suggestive of a third phase of distribution was observed, but this putative third phase was too close to the detection limit of the method used to allow its unequivocal identification. After oral administration of flunixin, plasma levels of the drug peaked within 30 minutes and then declined at rates which closely matched those after i/v administration of the drug. Flunixin was about 80% available after oral administration of the drug. Following i/v administration urinary levels of flunixin and its alkaline releasable metabolites peaked at about 200  $\mu$ g/m and were detectable in urine at about 100 ng/ml for up to 48 hours after dosing.

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